TRANSIENT PARALYSIS AS AN EPILEPTIC EQUIVALENT.[1]

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Transitory paralyses subsequent to epileptic seizures, whether such seizures are of the Jacksonian or ordinary type, are sufficiently well known to require little more than mention in this paper. Suffice it to say that they may be monoplegic or hemiplegic in variety, and may be due either to exhaustion or to inhibition. Other transitory paralyses related to epileptic attacks are of much less frequent occurrence, but at times occur, even some years previous to the onset of epileptic seizures; or, the epilepsy being established, they take place in the interval between the convulsions. Those which are observed before convulsions are seen at all, may be due to any one of a variety of causes; among which are syphilis, cerebral neoplasm, cerebral softening, general paralysis, etc. Of those occurring in the interval of epileptic convulsions, in illustration of which I here report a case, the cause seems to be, like that of the major condition, somewhat obscure.

H. is an intelligent unmarried female, 37 years of age, a native of the United States, and without any especial business. Her family history includes cases of degenerative mental disease, organic nervous disease, and tuberculosis; but no epilepsy, chorea or any form of convulsive disorder. She had convulsions when nine months old, and had them subsequently at intervals until three years of age. For four years she was free from seizures, but at seven years she had two convulsions in twelve months. Up to this time the attacks were mostly unilateral, involving the left side. At fourteen years of age menstruation started and the appearance of the menstrual flow was associated with general convulsion. From that time to the present she has had convulsive seizures at irregular intervals, although for about five years they have been greatly diminished in frequency and lessened in severity. The attacks are undoubtedly epileptic. She has never had any post-paroxysmal paralysis or paresis, or any abnormal mental condition. For the past two and one half years H. has had occasional attacks of paralysis involving the left arm and the left leg, either separately or together. The attacks last from a few seconds to two or three minutes; consciousness is never lost; they are preceded by prodromata in the form of paresthesia which involve the limbs to be paralyzed. When the paralysis is developed, whatever the patient is holding in the hand is dropped; or if she is standing and the leg is affected, she will sink to the floor. There is no aphasia, no amnesia, no involvement of the cranial nerves or of general sensibility. Control of the sphincters is perfect. After the spell is over the patient proceeds with whatever she was doing when it came on, and this occupation is sometimes not interrupted by the attack. The attacks occasionally take place at night, the patient being roused from sleep by the paresthesia. The paralyses never come on during or after a convulsion, but rather in the interval between the convulsive seizures.

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[1] Read before the Phila. Neurological Society, April 24, 1899.

Summarizing, we have a person, the victim of epilepsy, who has at times paresthesia followed by paralysis, the loss of power being transient, unaccompanied by loss of consciousness or memory, or by any involvement of the cranial nerves. No convulsive movements either precede or immediately succeed these attacks.

In searching for some explanation of these paralytic phenomena, I am utterly unable to find any satisfactory theory either as to their nature or as to their cause. Very few such cases are reported, and those which I have found I have embodied in this report.

Higier,[2] of Warsaw, recently reported the case of a boy who had epilepsy since infancy, although at the time of the report he had been free from convulsions for some time. At five and one half years he again had epileptic seizures, and associated with them were momentary attacks of paresthesia with paralysis of the right leg. Sometimes the weakness extended to the trunk muscles and to the right arm. If standing, the boy would fall, but would not lose consciousness. The attacks lasted ten or twenty seconds, were frequently repeated and were not accompanied by aphasia or mental confusion. The paralysis occurred during sleep, the boy being waked by the paresthesia. There were no hysterical stigmata.

The same author later cites[3] the case of a girl who had inflammation of the brain at three years of age, with convulsion and subsequent contracture of the left upper limb. In the last few months she has had in the intervals between epileptic seizures attacks of paralysis in the left lower and upper limbs, preceded by paresthesia, unaccompanied by loss of consciousness. The paroxysms last from ten to twenty-five seconds, during which time the left arm, usually contractured, is flaccid and its immobility disappears.

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[2] Higier, H., Neurolog. Centralbl., 1897, No. 4, p. 152.

[3] Higier, H., Deutsche Zeitschr. f. Nervenheilk., V. 14, 1899, Nos. 3 and 4, p. 325.

Pitres[4] tells of a man who had epilepsy from childhood, and who at intervals during the course of that disease had transient paralyses of the right upper limb, without preceding convulsion.

The case is recorded by Bouchaud[5] of a woman fifty-three years of age who had repeated attacks of fugacious brachial monoplegia during fifteen years. Finally epileptic seizures of the Jacksonian variety supervened and were associated with paralysis in the epileptic interval.

Wood,[6] years before the varieties of epileptic attacks were differentiated, claimed to have observed epileptic seizures which occurred "without loss of consciousness, with paralytic sensations and loss of power of particular limbs."

Whether or not these transient paralyses can be classed as attacks of petit mal, or whether we can consider them as actually taking the place of the epileptic seizures, is a question. Petit mal consists merely in some transient loss of consciousness and very little or no convulsive movement,[7] although occasionally we have only spasm without loss of consciousness.[8] In the case here reported there has never been the slightest loss of consciousness in connection with the paralyses, nor has there been the suggestion even of a spasm. This statement is based upon my personal observation of many of the attacks of paralysis, and upon the statements of the patient, who is a very intelligent woman despite her affliction. Certain it is, the paralyses do not exactly resemble petit mal; they do not follow the convulsive seizures; nor, on the other hand, do they immediately precede them. They occupy a separate and distinct position, in which they appear to be the substitutes of the convulsive attacks, or, as some have called them, the "equivalents" of epilepsy. Pitres[9] is one of the writers who has described these equivalents, and in commenting on them says: "It is less commonly known that epileptic patients may have without initiatory convulsion sudden transient paralyses, limited to a limb or extending throughout one side of the body. These transient paralyses appear suddenly in the midst of perfect health."

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[4] Pitres, A., Revue de Médecine, V. 8 ,1888, p. 609.

[5] Bouchaud, Journal de Neurologie, October 5, 1898, p. 392.

[6] Wood, G. B., "Treatise on the Practice of Medicine," 5th ed., 1858, p. 785.

[7] Hamilton, A. McL., "Syst. of Pract. Med.," ed. by Wm. Pepper, M.D., 1886, V. 5, p. 477.

[8] Gowers, W. R., "Epilepsy and Other Chronic Convulsive Diseases," 1885, p. 90.

Higier[10] regarded his two cases as instances of partial paralysis appearing as an epileptic equivalent, and employs arguments to support such a belief as follows:

1. In typical epilepsy the patient sometimes has attacks in which unconsciousness occurs, but no convulsion.

2. A brief paralytic period is observed after typical Jacksonian epileptic attacks, although the paralysis is not always proportionate to the convulsion. We also have partial epilepsy with simultaneous convulsion of one side and paralysis of the other.

3. Weakness of one limb may develop with, or precede, a Jacksonian convulsion.

4. In cases of partial sensory epilepsy loss of function of a sensory organ occasionally takes place, and sometimes rare cases of both motor and sensory inhibition are seen.

5. Ophthalmoplegic migraine is an undoubted epileptic equivalent, and here loss of function (ocular paralysis) is prominent.

6. Chorea has many clinical and pathological similarities to epilepsy, and of this a paralytic form[11] (chorea mollis s. paralytica) is seen, in which paralysis of one or both sides precedes the clonic stage.

While Higier's arguments are intended to prove the occurrence of these motor paralytic equivalents, it seems to me he rather relates them to post-paroxysmal paralyses, or, at least, his distinction between the two is not very clear. His object is evidently to show the frequency of the occurrence of paralysis in epilepsy and allied diseases.

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[9] Pitres, *op. cit.*

[10] Higier, *op. cit.*

[11] Gowers, W. R., Brit. Med. Jour., April 23, 1881, p. 636.

In studying the subject of transient paralyses one symptom group, recently so well studied by Taylor,[12] has impressed me as perhaps in some way related to these transient paralyses occurring in the epileptic intervals. This is the so-called family periodic paralysis. I do not assert that there is an absolute relationship between the two conditions, but some points of resemblance suggest such a possibility. These points of similarity are the influence of heredity; the recurrent character of the conditions; the occurrence of prodromata; the frequent nocturnal onset of the troubles; the retention of consciousness throughout the duration of the paralysis; the headache or slight indisposition following the attacks. The peculiar electrical changes in the muscles in cases of family periodic paralysis have not been observed in the transient paralyses of which my case is an example, nor are diminished reflexes noted. Both conditions are paroxysmal appearances of limited disturbance of the motor spheres, although of different duration. In either the loss of function may be a monoplegia,[13] [14] or a hemiparesis,[15] although usually in family periodic paralysis both lower extremities are involved. Mellus,[16] in the discussion of the case of family periodic paralysis presented before this society by Dr. John K. Mitchell some months ago, said that the history of the patient suggested to him some of the cases of post-paroxysmal paresis without epileptic paroxysms.

In looking for the cause of these transient paralyses occurring in the epileptic interval I find little to throw light upon the subject, or to explain how transient paralyses can replace or can substitute epileptic convulsions.

Grasset[17] considers the two conditions, paralysis and convulsion, quite independent of each other, hence, says Pitres,[18] "it is possible that they, the paralyses, are dependent upon a sudden and temporary impairment of the motor centers." But what is the cause of this sudden impairment? Jackson[19] says that repeated transient paralyses of similar local involvement are hard to explain, but he offers the suggestion of a small clot or brain softening from occlusion of a small vessel. Bouchard[20] ascribed the symptoms in his case to a deep-seated cerebral glioma, although the presence of a neoplasm is not well made out in the clinical history of his patient.

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[12] Taylor, E. W., Jour. Nerv. and Ment. Dis., V. 25, 1898, Nos. 9 and 10, pp. 637, 719.

[13] Bouchaud, *op. cit.*

[14] Hirsch, K., Deutsche med. Wochenschr., 1894, No. 32, p. 646.

[15] Burr, C. W., Univ. Med. Mag., Phila., 1892-3, p. 836.

[16] Mellus, E. L., Jour. Nerv. and Ment. Dis., 1899, Vol. 26, No. 3, p. 178.

[17] Grasset, J., et Rauzier, G., "Traite pratique des Maladies du Système nerveux," 4th ed., 1894, V. 1, p. 284.

The agency of auto-intoxication in the production of these motor paralytic equivalents is a theory not to be entirely set aside. Transitory paralyses, not necessarily related to epilepsy, sometimes occur as the result of toxemia. Achard and Levi[21] have reported cases of transitory paralyses of cardiac origin and relate them to those due to uremic intoxication and to the blood poisoning from hepatic disease. Van Gieson[22] believes that by the action of a toxic substance upon the lowly organized neuroglia cells and upon the mesoblastic connective tissue cells, increase of the neuroglia cells in the subcortical zones can readily be produced. Such a pathologic finding he has made in the brains of epileptics. Von Jaksch[23] found that epileptic attacks may be caused by auto-intoxication from urea or acetone, without the presence in the urine of sugar or albumin. Mairet[24] found that before an epileptic seizure the urine was hypertoxic, and that subsequently and in the interval it was hypotoxic. Cabitto[25] employed the sweat secreted in the prodromal and early stages of an epileptic convulsion and produced in rabbits similar convulsive conditions. No such result was obtained from the secretion of the decreasing stage or of the interval.

Making comparison again with family periodic paralysis, we find that Taylor[26] thinks that auto-intoxication is "by all means the most plausible working hypothesis" in seeking a cause for this symptom group, "the source of origin of the problematic poison being wholly obscure." This opinion is shared by Mills[27]. Goldflam[28] found the urine of a patient suffering with family periodic paralysis to be hypertoxic during the attack, and hypotoxic after recovery and in the interval. If auto-intoxication can cause, or be supposed to cause, transient paralyses of the family type, the origin of somewhat analogous loss of power might be considered possible of similar explanation, until at least, we know more of the exciting causes of epilepsy.

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[18] Pitres, *op. cit.*

[19] Jackson, J. Hughlings, Brain, Jan., 1881, V. 3, p. 437.

[20] Bouchard, *op. cit.*

[21] Achard and Levi, La Méd. moderne, 1897, 7, p. 656.

[22] Van Gieson, Ira, State Hosp. Bull., N. Y., 1896, V. 1, No. 4, pp. 470, 471, 472.

[23] Von Jaksch, R., Zeitschr. f. klin. Med., 1886, p. 199.

[24] Mairet, La France méd., 1897, No. 5, p. 70.

[25] Cabitto, C., Riv. Speriment. di Freniat., 1893, 27, p. 36. Abstract in Jour. Nerv. and Ment. Dis., 1898, V. 25.

[26] Taylor, *op. cit.*

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[27] Mills, C. K., Jour. Nerv. and Ment. Dis., 1899, V. 25, No. 3, p. 178.

[28] Goldflam, quoted by Van Gieson.

107. ZUR PATHOGENESE DER HEMICHOREA POSTAPOPLECTICA (Contribution to the Pathogenesis of Post-apoplectic Hemichorea). W. Muratow (Monatsschrift für Psychiatrie und Neurologie, Vol. 5 1899, p. 180).

Muratow reports two cases, one with necropsy, as a contribution to our knowledge of forced movements. They seem to show that a lesion of the fibers forming the anterior cerebellar peduncle and the nucleus ruber causes hemichorea or athetosis. He believes that Bonhoeffer has definitely proven that a lesion of these fibers produces such movements. Muratow's second case is evidence that hemichorea may result from a primary lesion of a cerebellar hemisphere and secondary degeneration of the anterior cerebellar peduncle. He believes that post-apoplectic forced movements indicate a disturbance of co-ordination in the functions of the cerebellum, basal ganglia and the cells of the anterior horns. Involvement of the cerebral cortex and of the pyramidal fibers cannot be excluded, but the cortex is involved only by means of the afferent tracts, and focal cortical lesions of the central convolutions do not produce such involuntary movements. He speaks, however, of certain forms of cortical forced movements which he does not separate sufficiently sharply from the subcortical forced movements. Athetoid and choreic forced movements are not associated with disturbance of the muscle-sense or cortical epilepsy. Muratow finds that a lesion of the nucleus ruber causes forced movements on the opposite side of the body; and one of the cerebellum, forced movements on the same side. Post hemiplegic tremor is a spinal symptom, and indicates an increased tonicity of the cells of the anterior horns. SPILLER.